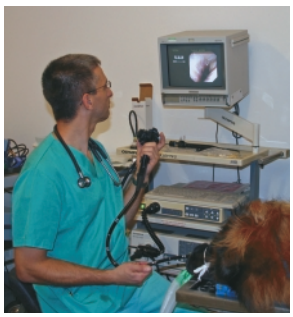

When investigating oesophageal disease, initial diagnostic efforts should concentrate on ruling out treatable primary diseases and identifying complications

Diagnosis and management of canine oesophageal disease and regurgitation

CLIVE ELWOOD

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DISORDERS associated with regurgitation and failure of oesophageal function can be difficult to define and frustrating to treat. Aspiration pneumonia is a common and potentially life-threatening sequela and, for this and other reasons, every effort must be made to define the disorder and institute appropriate management as early as possible. Often, various treatments may need to be tried and tested to determine which is most suitable for an individual patient. This article provides guidance on diagnostic approaches and treatment options for the clinician presented with a dog with suspected oesophageal disease.

THE NORMAL CANINE OESOPHAGUS

The normal canine oesophagus is a multilayered muscular tube that carries food and liquid from the pharynx to the stomach; it is composed of fibrous, muscular, submucosal and mucosal layers. There is skeletal muscle throughout the length of the oesophagus, the two spiralling layers of which cross obliquely at the dorsal and ventral lines such that the deep layer on one side is the superficial layer on the other. The cranial end of the oesophagus begins at the level of the axis (C2) and the caudal larynx, and the first muscle fibres blend with the cricopharyngeal muscle. The cervical portion courses to the left of the trachea. At the thoracic inlet, the thoracic

Potential causes of regurgitation

Anatomical
- Cardiac vascular ring anomaly
- Cricopharyngeal stenosis
- Cricopharyngeal achalasia
- Hiatal hernia
- Diverticulum
- Broncho-oesophageal fistula

Anomalous
- Gastro-oesophageal intussusception

Oesophagitis
- Traumatic
- Reflux (eg, hiatal hernia, poor sphincter function, general anaesthesia)
- Irritant (eg, dietary indiscretion, medications)
- Secondary (eg, megaoesophagus)

Obstructive
- Extraluminal
  - Mediastinal masses (eg, lymphoma)
  - Cervical masses
  - Retropharyngeal lymphadenopathy
- Mural
  - Neoplasia
  - *Spirocerca lupi* infection
  - Stricture (usually subsequent to oesophagitis)
- Luminal
  - Foreign body

Neuromuscular
- Idiopathic megaoesophagus (congenital/acquired)
- Myasthenia gravis
- Botulism
- Tetanus
- Myositis
- Myopathy (eg, dystrophin deficiency)
- Neuritis (eg, polyneuritis)
- Neuropathy (eg, bilateral vagal damage)
- Central (brainstem disorders such as neoplasia)
- Hypothyroidism
- Hypoadrenocorticism
- Toxicity (eg, lead, thallium, anticholinesterase)
- Dysautonomia
- Sialadenosis

segment begins to course dorsally so that it lies dorsal to the trachea at the bifurcation, where it crosses the caudal mediastinum to the oesophageal hiatus of the diaphragm. After passing the diaphragm, a short abdominal segment merges with the stomach. The positive pressure on the abdominal oesophagus, oesophageal muscle tone, gastric rugal folds, diaphragmatic muscles and the effects of gastric distension all contribute to the gastro-oesophageal sphincter, which prevents reflux of irritant gastric acid into the oesophagus.

Sensory and motor innervations are carried in branches of the 10th cranial (vagus) nerve and are essential for normal oesophageal function in dogs. Following the pharyngeal phase of swallowing, a bolus of ingesta enters the oesophagus and, as part of the swallowing reflex, is propelled aborally by primary peristalsis. If primary peristalsis fails, sensations of local oesophageal distension trigger a wave of secondary peristalsis. Sensory input also appears to be needed to propagate primary peristalsis. As the bolus reaches the distal oesophagus, there is reflex relaxation of the muscles of the gastro-oesophageal sphincter, which allows passage of the bolus into the stomach.

SIGNS OF OESOPHAGEAL DISEASE

Regurgitation of food or liquid is the most commonly recognised sign of oesophageal disease, although hypersalivation, anorexia, odynophagia (pain on eating), dysphagia, nasal discharge and coughing (as a result of aspiration pneumonia) can all be primary presenting signs. Regurgitation is the retrograde expulsion of ingesta from the oesophagus or pharynx. Unlike vomition, there are often no prodromal signs, no reflex abdominal activity ('heaving') and ingesta is returned passively under the influence of gravity. Salivation, gagging and extension of the neck may be seen with regurgitation. Usually, the ingesta returns as it was swallowed (unless there is some accompanying gastric reflux) and, in the case of food, may be in the form of a tubular cast with a coating of saliva. Regurgitated saliva is often described as having the consistency of 'whisked egg whites'. Regurgitation can occur soon after ingestion, during swallowing in some cases (eg, cricopharyngeal stenosis), or many hours afterwards. Thus, timing after ingestion cannot be used to distinguish between vomiting and regurgitation.

DIFFERENTIAL DIAGNOSIS OF OESOPHAGEAL DISEASE

Any process that interferes with normal oesophageal function (see box on page 14) can cause signs of oesophageal disease. Intraluminal or extraluminal obstructions prevent the normal passage of ingesta, neuromuscular diseases cause muscle weakness and atrophy, and sensory deficits can prevent normal peristalsis. A common, but not inevitable, consequence of many of these situations is megaoesophagus, which results in a dilated, weak and hypomotile oesophagus. Failure of oesophageal function leads to pooling of ingesta and, possibly, regurgitation. As regurgitation is not associated with reflex closure of the larynx and protection of the airway, aspiration pneumonia is a common, and potentially life-threatening, consequence of oesophageal disease.

INITIAL INVESTIGATION

SIGNALMENT

In puppies, signs of oesophageal disease may manifest at an early stage with oronasal reflux of milk and, possibly, pneumonia. More typically, signs are noticed at weaning when it becomes clear that solid food is being regurgitated. At this age, the major considerations are cardiac vascular ring anomalies causing extraluminal compression of the oesophagus and congenital idiopathic megaoesophagus.

Breed associations include a tendency for terriers (especially West Highland white, Scottish and Cairn terriers) to present with obstructing foreign bodies, and an increased risk of megaoesophagus in German shepherd dogs, golden retrievers and Irish setters.

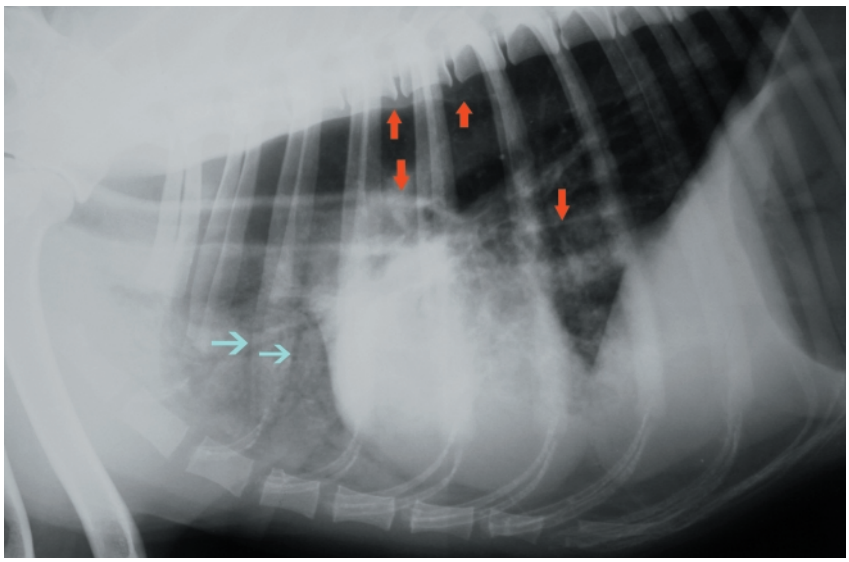
HISTORY

The history should be used to distinguish between regurgitation and vomition by seeking an exact description of events. Occasionally (eg, with sliding hiatal hernias), both vomiting and regurgitation may be described. Systemic signs, such as malaise, lethargy, inappetence and weight loss, might indicate a primary systemic illness or, alternatively, complications such as pneumonia (in which case coughing and dyspnoea may also be a feature). Muscle weakness, exercise intolerance, ataxia and/or focal neurological deficits might indicate generalised neuromuscular disease. Dysuria and constipation can be associated with dysautonomia. Exposure to infectious agents (eg, while travelling) or toxins such as lead or botulinum (eg, via contaminated feed) may also be pertinent. Botulism is typically associated with packs of hunting dogs fed fresh meat. Evidence of foreign body exposure could indicate obstruction. Signs developing after general anaesthesia might indicate a reflux oesophagitis and stricture formation. The animal's eating behaviour, including appetite, speed of ingestion, ability to swallow and preference for liquid or solid food, can give clues about the likely aetiology. Painful conditions, such as oesophagitis, may be associated with a reluctance to eat, and swallowing function may be compromised where there is more generalised neuromuscular disease.

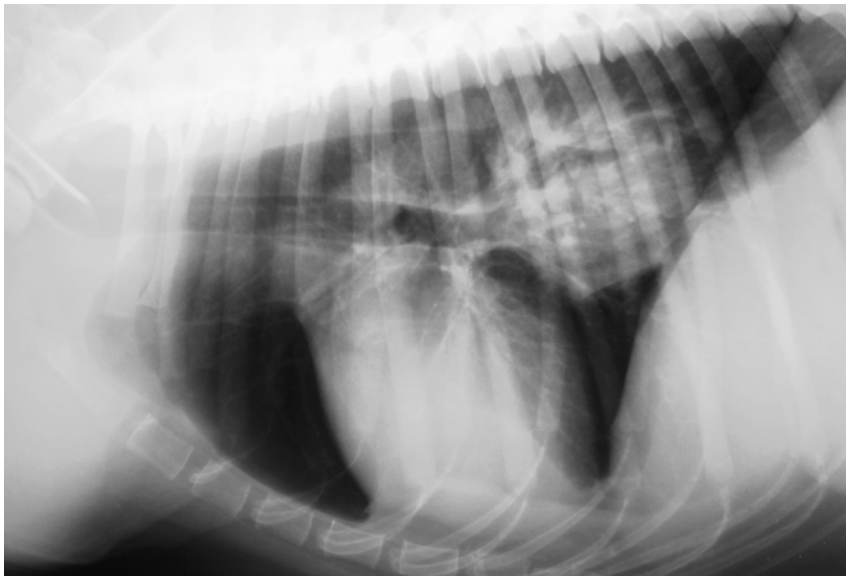
GENERAL PHYSICAL EXAMINATION

Any evidence of systemic disease, such as muscle weakness (eg, a tendency to sit down) or muscle pain as indicators of generalised muscle disease, should be noted. Fever, coughing, dyspnoea and signs of hyperdynamic/septic shock can be indicators of secondary aspiration pneumonia. A dry nose and mouth, distended bladder, palpable faecal boluses and dry rectal membranes might be seen with dysautonomia and, possibly, hypoadrenocorticism. Hypersalivation can be seen when swallowing function is affected, but also when there is associated nausea. Palpation of the cervical oesophagus can reveal trapped ingesta indicating megaoesophagus and a failure to pass ingesta into the thoracic oesophagus and stomach. Cervical palpation may also indicate the presence of obstructive mass lesions. Cranial mediastinal masses can, on occasion, cause obstruction to venous drainage, associated jugular venous distension and, potentially, oedema of the head (cranial vena cava syndrome).

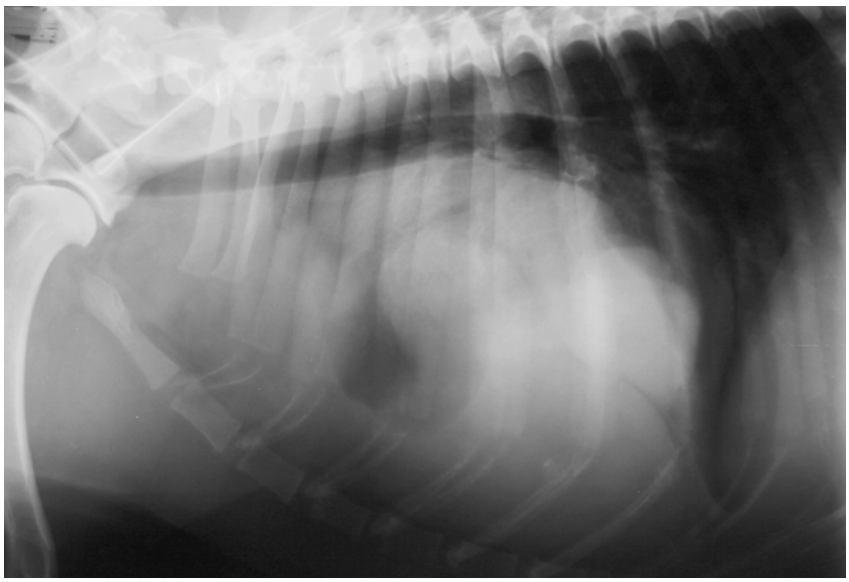
Findings on history taking or physical examination may indicate the need for a full neurological examination, which should aim to localise disease processes to



Lateral thoracic radiograph showing megaoesophagus (red arrows) and evidence of aspiration pneumonia (consolidated ventral lung lobes and air bronchograms; blue arrows) in a four-year-old Border collie with myasthenia gravis



Lateral thoracic radiograph showing an oesophageal foreign body in a four-year-old Border collie. The foreign body (a dog chew) was grasped and extracted via the mouth using a rigid endoscope



Lateral thoracic radiograph showing an anterior mediastinal mass in a 10-year-old akita/labrador cross. A thymoma was diagnosed and surgically removed. A mass such as this could cause regurgitation via direct compression of the oesophagus or via thymoma-associated myasthenia gravis

the central nervous system, nerves, neuromuscular junction or muscles and, ideally, indicate whether the disease is likely to be diffuse or focal.

AIDS TO DIAGNOSIS

Haematology and biochemistry

A haematology panel is indicated to screen for inflammatory diseases, including infections such as toxoplasmosis and neosporosis, and for aspiration pneumonia. Primary endocrinopathies (eg, hypothyroidism and hypoadrenocorticism) may be associated with anaemia. A biochemistry panel should be examined for evidence of endocrinopathies (eg, sodium/potassium abnormalities, hypercholesterolaemia) and for signs of muscle damage (eg, increased creatine kinase and aspartate aminotransferase). Hypokalaemia and hypochloraemia can indicate loss of gastric acid through vomition and thus help to distinguish between vomiting and regurgitation. In the absence of clear evidence of another disease process, an antiacetylcholine receptor antibody titre is indicated to screen for focal myasthenia gravis. The level of serum thyroxine (or free thyroxine) and thyroid-stimulating hormone should be determined to screen for hypothyroidism, although care should always be taken when interpreting results because of the risk of 'euthyroid sick syndrome'. An adrenocorticotropic hormone (ACTH) stimulation test should be used to rule out hypoadrenocorticism; ensure that no corticosteroids have been administered prior to the test. If lead poisoning is considered possible, blood lead concentrations can be measured. Serology for toxoplasmosis and neosporosis is indicated if diffuse neuromuscular disease is suspected, especially if there is clinical or haematological evidence of infection/inflammation.

Imaging

Plain radiographic examination is useful to screen for oesophageal dilation, foreign bodies and aspiration pneumonia. Thymomas can trigger myasthenia gravis, and may be suspected from radiographs showing an anterior thoracic mass and megaoesophagus. As anaesthesia and sedation can cause relaxation of the oesophagus, and this can mimic megaoesophagus on radiographs, attempts should always be made to obtain images from conscious, unsedated patients. Standing conscious lateral thoracic radiography is useful if facilities allow, and may permit imaging of patients that would not otherwise lie restrained for conscious images. When an oesophageal tear is suspected (eg, due to a foreign body), radiographic signs of pneumomediastinum may be seen. Rapid intervention is required in such cases because of the risk of severe sepsis, which can be difficult to treat.

If plain radiographs are unhelpful, contrast radiographic examination can be useful. Administration of liquid contrast media can delineate the oesophagus and, potentially, the position of the cardia, and can help to confirm the presence of radiolucent foreign bodies. Note that, if regurgitation and aspiration of contrast or oesophageal tears are considered possible, water-soluble contrast media should be used to prevent insoluble barium entering the airways or mediastinum and triggering a chronic inflammatory response. A 'barium meal' (feeding a mixture of food and barium) can also be a useful means of detecting oesophageal disease, including megaoesophagus, oesophageal narrowing (eg, due to vascular ring

Set-up for obtaining standing conscious thoracic radiographs

anomalies), strictures and extraluminal compression; however, care should be taken to minimise the risk of aspiration by avoiding this technique in very debilitated patients or where swallowing or laryngeal/pharyngeal function appears to be compromised.

Fluoroscopy is an excellent means of assessing swallowing function, oesophageal motility, gastro-oesophageal reflux and the functional anatomy of the gastro-oesophageal sphincter. It is the preferred contrast radiographic technique for the study of swallowing and oesophageal function in the author's practice. During a swallowing study, barium-coated food is offered and swallowing function is imaged for a period sufficient to judge the effectiveness of swallowing; this is followed by imaging the length of the oesophagus as boluses of the barium meal pass along it, and then the gastro-oesophageal sphincter region to allow identification of reflux and, potentially, sliding hiatal herniation, which can be very transient. Recording of the study allows effective review. Abnormalities that can be identified include:

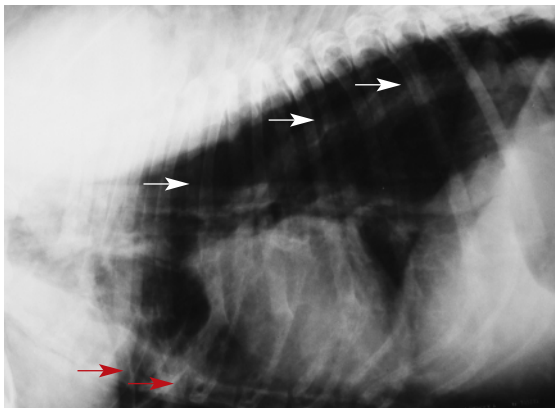
- An inability to relax the proximal oesophagus, leading to difficulty in swallowing;
- Poor oesophageal motility, leading to slow or ineffective passage of swallowed boluses;
- Oesophageal dilation and compression, reflux and adherence of contrast to the mucosa, which can be a sign of oesophagitis and failure of sphincter function.

Fluoroscopy appears to be a much more sensitive technique for identifying oesophageal dysfunction than standard plain or contrast radiography. Fluoroscopic examination of swallowing before and after the administration of edrophonium could be used to screen for myasthenia gravis with oesophageal involvement, but the use of this technique is not reported in the veterinary literature.

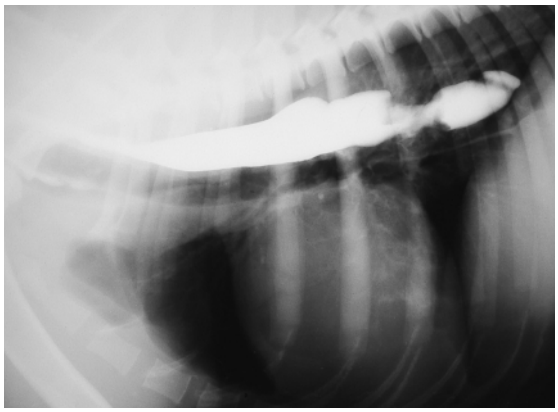
Other, advanced imaging techniques such as computed tomography and magnetic resonance imaging may be useful in certain cases, such as for the assessment of thoracic masses and where an intracranial lesion is suspected.

Endoscopy

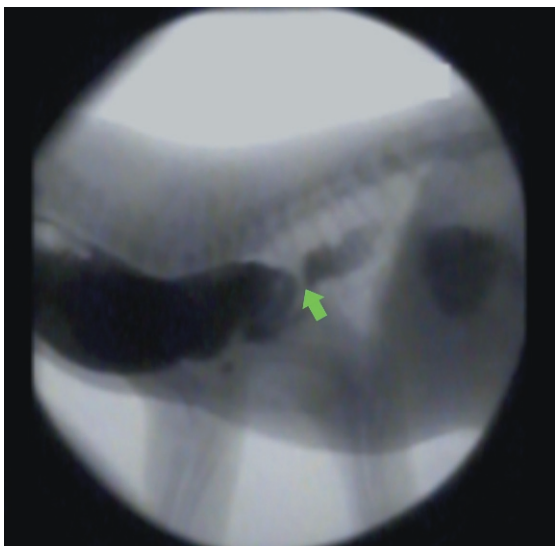
Both rigid and flexible endoscopic examinations can be useful to identify mucosal lesions, foreign bodies, stric-



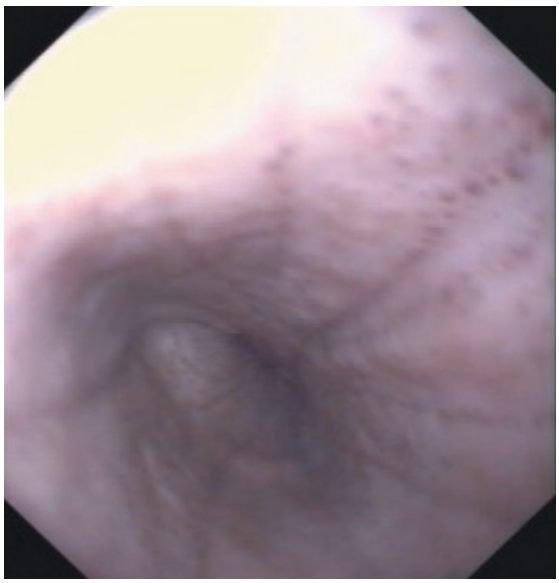
Pneumomediastinum can potentially result from oesophageal perforation. Note the mediastinal vessels (white arrows) and associated subcutaneous emphysema (red arrows)



Positive contrast oesophagogram ('barium swallow') in a dog with megaoesophagus. Note that sedation and anaesthesia can cause oesophageal relaxation, which could mimic these findings



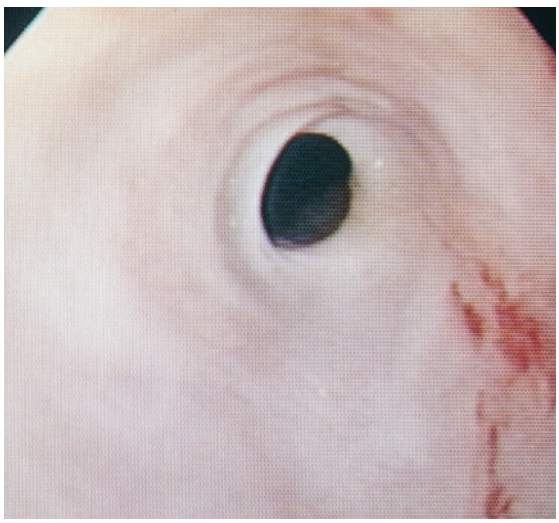
(left) A seven-week-old Norfolk terrier puppy undergoing a positive contrast fluoroscopic study to investigate regurgitation noticed at weaning. Note that manual restraint is avoided. (right) The resulting oesophagogram shows an oesophageal pouch with constriction over the heart base (arrow) and more normal oesophageal dimensions distally; these findings are consistent with a vascular ring anomaly. The puppy was successfully treated by surgical division of a persistent right aortic arch



Normal endoscopic appearance of the distal oesophagus and cardiac sphincter in a dog

Endoscopic view showing a fish hook foreign body embedded in the oesophageal mucosa. The associated line can be seen trailing towards the stomach. Rigid or flexible endoscopic retrieval may be possible, but care must be taken to avoid further snagging of the mucosa or damage to the endoscope, and to ensure that no perforations occur

A bone foreign body being removed from the oesophagus using a flexible endoscope and rigid grasping forceps. If a fragment of bone, such as this, cannot be removed via the mouth, it can sometimes be pushed into the stomach and left to digest



Endoscopic appearance of an oesophageal stricture with associated oesophagitis

tures and compressions, and to allow therapeutic and biopsy procedures. True megaoesophagus and early oesophageal dysfunction are not easy to identify because the image obtained with a flexible endoscope cannot be effectively scaled, but associated (primary or secondary) oesophagitis may be apparent as bleeding or streaking of the mucosa, prominent submucosal blood vessels and increased granularity or friability of the mucosa. It is often extremely difficult to obtain effective perendoscopic oesophageal mucosal biopsies because of the toughness of the oesophageal mucosa; the use of serrated biopsy forceps with a central spike to anchor the forceps to the mucosal surface may improve procurement.

When generalised or focal muscle disease is suspected, further investigation can include electromyography to screen for muscle damage and muscle biopsy to help define the pathology. Biopsies should be obtained from an affected area of muscle where sampling is not likely to cause problems, such as the anterior tibial muscle, or the gracilis or dorsal lumbar muscles. Muscle biopsy samples should be obtained in sufficient quantity, processed accordingly and submitted to a laboratory (ideally one that is experienced in staining and evaluating diseased muscle).

MANAGEMENT OF OESOPHAGEAL DISEASE

Clinical management of oesophageal disease, particularly idiopathic megaoesophagus, can be difficult and frustrating, with some cases doing unexpectedly well and others faring worse than hoped. A combination of treatments may be required and often various regimens need to be tried and tested to find out which is optimal for an individual patient. If a primary disease process can be identified, this should be treated. Therapy may be relatively straightforward and associated with an expected return to normal oesophageal function (eg, in patients with hypoadrenocorticism). However, identification and treatment of the primary disease is not always expected to lead to resolution of the oesophageal signs and continued symptomatic therapy may be necessary (eg, in cases of myositis).

Oesophagitis

Oesophagitis may be a secondary consequence of oesophageal disease (due to the oesophagus failing to empty effectively, thus resulting in stagnation and infection of ingesta) or a primary cause of oesophageal motility disorders. Where a primary trigger for oesophagitis is identified, this should be treated. General treatment approaches include the administration of sucralfate suspension, which acts as a chemical bandage coating ulcerated/exposed mucosa and promoting re-epithelialisation. Increasing the gastric pH with the use of H_2-antagonists is appropriate if there is significant gastric reflux. Ranitidine is available as a syrup, which may improve the passage of the drug into the stomach for absorption, and is also available for parenteral administration. Proton-pump inhibitors (PPIs) such as omeprazole are preferred in humans for the treatment of reflux oesophagitis, but are not available in parenteral forms for dogs and have a delayed onset of action, which makes them less useful for acute disease where rapid effects are desired. PPIs are suitable for administration for four to six weeks but, if given for longer, increase the risk of gastric infection and other complications because of chronic hypoacidity.

Myasthenia gravis

The treatment of myasthenia gravis and combined megaoesophagus is particularly difficult. Standard treatment regimens for myasthenia gravis include acetylcholine esterase inhibitors (typically corticosteroids), with or without immunosuppression. Due to regurgitation, oral medication may be unpredictable, so injectable treatments (eg, neostigmine and dexamethasone) may be preferred. Major side effects of acetylcholine esterase inhibitors are hypersalivation, which can challenge an already hypofunctional oesophagus, and vomiting, which may lead to or exacerbate aspiration pneumonia. Careful upwards titration of the dose of inhibitor should be performed but, in some cases, it is not possible to achieve effective control of weakness before side effects become problematic. Typically, megaoesophagus associated with myasthenia gravis may not resolve for a number of weeks, if at all. In such cases, immunosuppression may be indicated if other treatment is ineffective, but this increases the risk of severe complications such as aspiration pneumonia and should ideally be avoided if pneumonia is already present.

Foreign body obstructions

Oesophageal foreign body obstructions are emergencies and should be removed as soon as it is safe to do so. Many objects can obstruct the oesophagus, but bone obstructions are commonly seen. Objects with sharp edges may perforate the oesophageal wall, and the risk of this, and other complications such as stricture formation and aspiration pneumonia, will increase the longer the object remains in situ. While non-surgical management of oesophageal foreign body obstructions is preferred, the owner and clinician should be prepared for the need for surgical intervention, whether to retrieve an object pushed into the stomach, to surgically remove an otherwise irretrievable obstruction or, potentially, to deal with any perforations. Non-surgical removal may be facilitated by rigid and/or flexible endoscopy with a variety of instrumentation. Fluoroscopy is also useful. After removal, water-soluble contrast media may be administered prior to further radiography to check for perforations or tears. Due to the technical issues associated with managing this condition, the high-risk anaesthesia involved and the potential need for thoracic surgery, many cases of oesophageal foreign body obstruction may best be referred to an appropriate institution.

Strictures

Oesophageal strictures can be single or multiple, thin membranous structures or more extensive in length. Strictures can be caused by exogenous irritants (eg, inappropriate ingestion of caustic substances) or reflux of gastric acid (most commonly during general anaesthesia), or may develop subsequent to foreign body obstruction. Strictures are managed by dilation, either using bougies or, more commonly, balloons inflated intraluminally. Balloons designed for dilation of human colonic strictures can be passed across the stricture under endoscopic guidance and inflated with water to an appropriate pressure with a dedicated inflator. Balloons can alternatively be improvised from endotracheal tubes, allowing gradual dilation with the use of progressively

Feeding management for oesophageal disease

Most diseases that are associated with regurgitation require feeding management. As dogs normally pass food into the stomach without the assistance of gravity, significant benefit can be obtained by raising food and water bowls so that the dog's head is higher than the stomach when eating and drinking. This can be achieved by using special food stands that are commercially available; alternatively, the placement of food on steps or stairs, or even on a work surface, can be effective, assuming the patient has sufficient strength to hold itself upright. Owners can also be advised to hold the patient with the forelimbs raised for approximately five minutes after a meal.

Ingestion of food may be improved by feeding it at an altered consistency. The preferred consistency for a given case may be difficult to predict so a variety of consistencies, ranging from liquidised food to boluses of firm food, should be tried. Food fed as moulded balls may reduce the risk of aspiration if there is associated dysphagia. In principle, where there is a degree of obstruction (eg, a stricture) or a suspected muscle weakness (eg, myopathy), soft to liquid food may be better tolerated; conversely, where an early sensory deficit is suspected (eg, idiopathic megaoesophagus), firm food may be preferable as it is likely to be sensed in the oesophagus more readily.

Holding animals upright after eating can help the passage of food into the stomach in cases of oesophageal dysfunction

In some circumstances (eg, prior to surgical management in a debilitated animal), the oesophagus may need to be bypassed for short-term nutritional support; this may be achieved by surgical or, if appropriate, endoscopic placement of a gastrostomy tube. If the oesophageal disorder is permanent and oral alimentation is not possible, placement of a permanent gastrostomy feeding tube can be considered; the success rate of this approach is not documented.

Balloon dilation catheter and inflator that may be used to stretch oesophageal strictures. To minimise the risk of tearing, care should be exercised to ensure that the pressure and duration of inflation is not excessive

bigger tubes. Effective dilation can be confirmed by endoscopic evaluation or via simultaneous fluoroscopy. After dilation, the oesophagus should be re-evaluated endoscopically to ensure all strictures have been treated; corticosteroids can be given at antifibrotic doses for four to six weeks and 'chunky' food should be fed to promote continual stretching of the stricture as healing occurs. Repeated stretching procedures may be necessary to achieve a successful outcome. Oesophageal tearing is a potential complication.

Vascular ring anomalies and other surgical conditions

Surgery is indicated for the management of certain vascular ring anomalies, such as division of a persistent right aortic arch, hiatal hernias and other anatomical abnormalities. Such surgery is technically demanding and often requires entry into the chest cavity; thus, these should be considered specialist procedures.

SUMMARY

When presented with a dog showing regurgitation and/or other signs of oesophageal disease, initial diagnostic efforts should concentrate on ruling out treatable primary diseases and identifying complications such as aspiration pneumonia. Treatable problems should be managed aggressively while further investigation continues to fully define the process. Where the primary problem is untreatable, symptomatic therapy can significantly improve an animal's quality of life, although outcomes are unpredictable and sometimes disappointing.

Further reading

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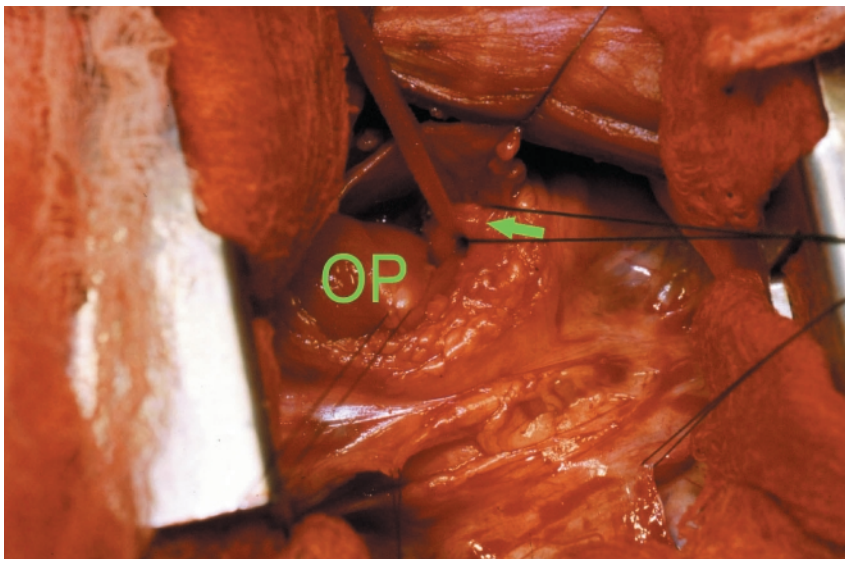
Management of aspiration pneumonia

Aspiration pneumonia is a common and sometimes life-threatening consequence of regurgitation. Aggressive management is appropriate in even moderate cases and should include:

- Judicious intravenous fluid therapy (particularly if the animal is not drinking and/or dehydrated) to treat shock/sepsis and increase the liquidity of secretions in the airways
- Broad-spectrum antibiotics (most pneumonias will be polymicrobial) such as a combination of a cephalosporin and fluoroquinolone, given intravenously if necessary, and, ideally, after a tracheal wash to determine the actual organisms involved and their antibiotic sensitivities
- Physiotherapy, specifically coupage, at regular intervals throughout the day

Pain relief for associated pleuritis should be considered. Adjunctive treatments include the use of bronchodilators, mucolytics and nebulisation therapy. In severe cases, airway suction, supplemental oxygen and mechanical ventilation may all be indicated.



Surgical management of a vascular ring anomaly via a left lateral thoracotomy. The aberrant vessel (arrow) can be seen as it courses dorsal to the oesophagus, causing constriction and an oesophageal pouch (OP) cranially. Picture, Dr Carolyn Burton

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